



# Role of thermal factors on aerobic capacity improvements with endurance training

**AD-A268 671**



2

ANDREW J. YOUNG, MICHAEL N. SAWKA, MARK D. QUIGLEY, BRUCE S. CADARETTE, P. DARRELL NEUFER, RICHARD C. DENNIS, AND C. ROBERT VALERI  
*US Army Research Institute of Environmental Medicine, Natick 01760-5007; and Naval Blood Research Laboratory, Boston University Medical Center, Boston, Massachusetts 02118*

YOUNG, ANDREW J., MICHAEL N. SAWKA, MARK D. QUIGLEY, BRUCE S. CADARETTE, P. DARRELL NEUFER, RICHARD C. DENNIS, AND C. ROBERT VALERI. *Role of thermal factors on aerobic capacity improvements with endurance training*. J. Appl. Physiol. 75(1): 49-54, 1993. — This investigation studied the importance of the rise in body temperature during exercise for aerobic capacity adaptations produced by endurance training. The approach used was to compare training effects produced by subjects exercising in hot (35°C) water vs. cold (20°C) water. Hot water was used to potentiate, and cold water to blunt, the rise in body temperature during exercise. Eighteen young men trained by cycle-ergometer exercise at 60% of maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) while immersed to the neck in either hot (HWT,  $n = 9$ ) or cold (CWT,  $n = 9$ ) water for 60 min, 5 days/wk, for 8 wk. Before and after training,  $\dot{V}O_{2\max}$ , erythrocyte volume, plasma volume, and vastus lateralis citrate synthase activity were measured. Training increased ( $P < 0.01$ )  $\dot{V}O_{2\max}$  by 13%, with no difference between HWT and CWT in the magnitude of the effect. Erythrocyte volume increased 4% ( $P < 0.01$ ) with training, with no difference between HWT and CWT in the magnitude of the effect. Plasma volume remained unchanged by training in both the HWT and CWT groups. Last, vastus lateralis citrate synthase activity increased by 38% with training, but there was no difference between HWT and CWT in the training effect. Thus, exercise-induced body temperature elevations are not an important stimulus for the aerobic adaptations to moderate-intensity endurance training.

aerobic exercise adaptations; physical fitness; body temperature regulation; maximal oxygen uptake; erythrocyte volume; citrate synthase

THE INTERACTION BETWEEN PHYSICAL fitness and thermoregulatory responses of humans has been the subject of considerable research. Previous investigations have studied the influence of physical fitness and/or exercise training on thermoregulatory responses to environmental heat (see Ref. 3 for a review) and cold (1, 2, 6, 16) stress. However, studies to determine how repeated thermal stress (heat or cold) and the associated thermal strain would affect responses to physical fitness training programs have not been reported. During exercise in hot water, body temperatures rise significantly, whereas the rise in body temperatures is blunted or eliminated during exercise in cold water (13, 14, 19). If the rise in body temperature during exercise and/or the associated thermoregulatory responses contribute to the stimulus for adaptations usually observed during endurance training,

then training in hot water might produce different adaptations than training in cold water.

For example, accumulation of muscle metabolites during exercise is more pronounced in hot than cool environmental conditions, possibly as a result of effects of elevated muscle temperature or a redistribution of blood flow from muscle to skin on rates of metabolic reactions (27). It has been theorized that one stimulus for training-induced adaptations in muscle metabolism is the alteration in intracellular homeostasis that occurs during muscular activity as cellular metabolism generates energy to sustain the contractile process (12). Therefore, during exercise in hot water a more pronounced alteration in intracellular homeostasis compared with exercise in cold water might augment the stimulus for metabolic adaptations to training. Also, whereas cardiac output during exercise at a given oxygen uptake ( $\dot{V}O_2$ ) is the same in cold and hot water, heart rate is lower, but stroke volume higher, during exercise in cold than hot water (17). These differences could cause adaptations in cardiac responses to exercise that differ from endurance training in hot vs. cold water. Furthermore, endurance training reportedly results in an increase in blood volume due to an increase in plasma and erythrocyte volume (7). Hypervolemia and plasma volume expansion have also been observed to result from repeated heat exposure, and the effect induced differs when exposures are passive (i.e., resting) vs. active, indicating that separate thermal and exercise factors regulated hypervolemic adaptations (8). Thus training in hot water could affect vascular volumes differently than training in cold water. Any or all of the aforementioned differences might result in differences in the improvement in maximal oxygen uptake ( $\dot{V}O_{2\max}$ ) with training in cold vs. hot water.

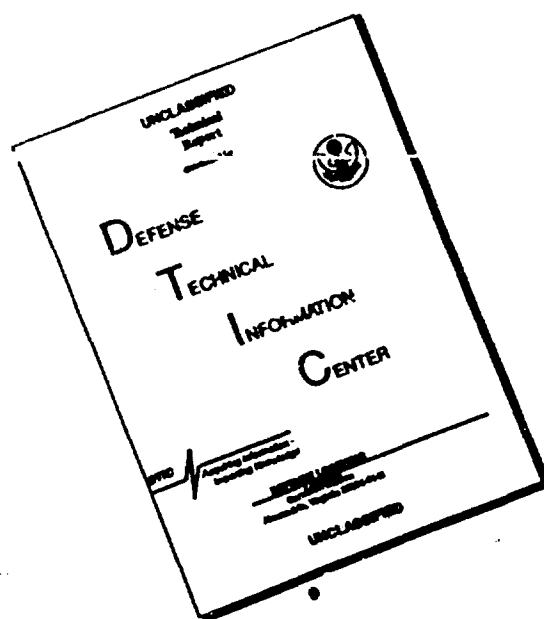
Thus this investigation studied the role of thermal factors for stimulating adaptations to endurance training. The effects of endurance exercise training in hot vs. cold water were compared. Hot (35°C) water was used to accentuate and cold (20°C) water was used to blunt the rise in body temperature during exercise training.

## METHODS

*Subjects and experimental design.* Eighteen healthy young male soldiers volunteered to participate after being informed of the requirements and possible risks associated with this research. Although all participated in

93 8 24 077

# DISCLAIMER NOTICE



**THIS DOCUMENT IS BEST  
QUALITY AVAILABLE. THE COPY  
FURNISHED TO DTIC CONTAINED  
A SIGNIFICANT NUMBER OF  
PAGES WHICH DO NOT  
REPRODUCE LEGIBLY.**

TABLE 1. Characteristics of subjects before training

|     | Age,<br>yr        | Height,<br>cm        | Mass,<br>kg       | $\dot{V}O_{2\max}$ ,<br>ml · kg <sup>-1</sup> · min <sup>-1</sup> |
|-----|-------------------|----------------------|-------------------|---|
| CWT | 20 ± 1<br>(18–26) | 176 ± 2<br>(168–187) | 77 ± 4<br>(64–99) | 46.0 ± 1.4<br>(40.6–54.7)   |
| HWT | 20 ± 1<br>(18–24) | 173 ± 2<br>(166–185) | 72 ± 4<br>(59–96) | 48.3 ± 2.0<br>(39.0–58.1)   |

Values are means ± SE with range in parentheses for 9 men per group. CWT, cold water training group; HWT, hot water training group.  $\dot{V}O_{2\max}$ , maximal oxygen uptake.

physical training programs required of all Army personnel before beginning this study, their participation was at the minimal level demanded and none was considered highly trained. This study was conducted in Natick, MA, during the winter months (January through March), when decay and induction of natural heat acclimatization were expected to be minimal and the usual New England weather conditions tend to discourage strenuous physical activity beyond that prescribed by the protocol.

Initially, the subjects' body composition and aerobic fitness as indicated by  $\dot{V}O_{2\max}$  were assessed. Erythrocyte volume, plasma volume, and vastus lateralis citrate synthase activity were also measured. Also, before the subjects began the training program, muscle temperatures were measured before and after 60 min of exercise in 35°C water, at the exercise intensity to be used during the 1st wk of training.

The 18 subjects were then divided into two equally sized groups, one designated the hot water training group (HWT) and the other designated the cold water training group (CWT). The five subjects exhibiting the highest postexercise muscle temperatures during the 60-min submaximal exercise bout in 35°C water were assigned to the HWT, and the five subjects exhibiting the lowest postexercise muscle temperatures during that bout were assigned to the CWT. The remaining subjects were assigned to the groups in such a way as to match the two groups (no significant differences) for  $\dot{V}O_{2\max}$ , height, mass, percent body fat, and age. The rationale for assigning subjects in this way was to create two groups whose muscle temperature response to exercise in hot water was significantly different (HWT = 39.2 ± 0.1°C, CWT = 38.8 ± 0.1°C;  $P < 0.05$ ) but whose initial fitness level and body composition were similar. It was thought that this would allow an opportunity to investigate whether the rise in muscle temperature during exercise was one of the factors influencing training-induced adaptations. Table 1 shows the average age, height, mass, and  $\dot{V}O_{2\max}$  for the two groups.

After completing the pretraining measurements, the subjects began the 8-wk endurance training program. During the 4th wk of training and again after completion of the 8th wk of training, measurements of  $\dot{V}O_{2\max}$ , erythrocyte volume, and plasma volume were repeated. Vastus lateralis citrate synthase activity was measured again after the 8th wk of training was completed.

Training consisted of exercise for 60 min/day, 5 days/wk, for 8 consecutive wk. Training was generally scheduled for Monday through Friday, with missed sessions (illness, etc.) made up on Saturday and/or Sunday. The

subjects exercised by pedaling a cycle ergometer modified for use in water (25) while seated upright and immersed to the neck in either 35°C (HWT) or 20°C (CWT) water. The exercise was performed in a 36,000-liter immersion pool, in which the water was continuously stirred by air bubbled from the bottom, and water temperature was maintained within ±0.3°C of the desired value. During each training session,  $\dot{V}O_2$  was measured at the 15th min of exercise; heart rate and rectal temperature ( $T_{re}$ ) were measured at every 10th min. Pedaling rate was 40 rpm.

It was desired that the subjects exercise at an intensity corresponding to 60%  $\dot{V}O_{2\max}$  throughout the entire 8-wk program. During the initial training session, subjects exercised at an intensity that had previously been determined (empirically) to elicit ~60%  $\dot{V}O_{2\max}$ . To offset the expected improvement in  $\dot{V}O_{2\max}$  with training and maintain the relative exercise intensity constant throughout the training program, it was necessary to periodically adjust (increase) the absolute intensity at which the subjects were exercising. This was accomplished by establishing the heart rate during exercise on the 1st day of training as a target heart rate. Whenever the exercise intensity was insufficient to elicit heart rate equal to the target on two consecutive training sessions, the intensity for the next session was increased.

**Experimental procedures.** The  $\dot{V}O_{2\max}$  determinations were made by using a discontinuous progressive intensity protocol, in which the subject exercised on a cycle ergometer (Monark) on land with an air temperature of ~23°C. The protocol for determining the  $\dot{V}O_{2\max}$  has been described in detail elsewhere (15, 18), and the criterion for accepting a  $\dot{V}O_2$  value as maximal was that an increase in power output of 15 W resulted in an increase of  $\dot{V}O_2$  of <75 ml/min.

An automated system (Sensormedics Horizon Metabolic Measurement Cart) that employs open-circuit spirometry was used to measure  $\dot{V}O_2$  and associated respiratory exchange parameters during consecutive 15-s intervals throughout the maximal exercise tests and during consecutive 1-min intervals from the 10th to the 13th min of each training session. During all exercise, the electrocardiogram (ECG) obtained from chest electrodes (CM-5 placement) and radiotelemetered to an oscilloscope-cardiotachometer (Hewlett-Packard) was continuously monitored. During maximal exercise testing, the heart rate was obtained from the ECG recorded during the final 30 s of each bout of exercise; the highest heart rate observed during the maximal exercise test was recorded as maximal heart rate. During the training sessions, heart rates were obtained from the ECG during the last 30 s of each 10-min interval. A thermistor inserted 10 cm past the anal sphincter was used to measure  $T_{re}$ , which was recorded during the last 30 s of each 10-min interval. Muscle temperature before and after the submaximal exercise bout in hot water was measured by using a thermocouple inside a 23-gauge hypodermic needle that was inserted 25 mm into the subjects' vastus lateralis.

Erythrocyte and plasma volumes were measured after 18–20 h recovery from previous exercise. The subjects were normally hydrated, and procedures were always

performed in the morning at the same time, with subjects having refrained from eating and drinking since the previous midnight. Measurements were made while the subjects were in the supine position and had rested quietly for 30 min. Erythrocyte volume was measured by using the radioactive chromium- ( $^{51}\text{Cr}$ ) labeled erythrocyte method and plasma volume by using the radioactive iodine- ( $^{125}\text{I}$ ) labeled albumin method, which have been recently described in detail elsewhere (24). Total blood volume was calculated by summing the measured erythrocyte and plasma volumes. On two occasions separated by 48 h before the subjects began training and two additional occasions after they completed 8 wk of training, plasma was separated from venous blood samples obtained from the subjects after 30 min of seated rest, and plasma protein concentration was measured by using refractometry (American Optical). Total circulating protein was calculated by multiplying plasma volume (measured the same week) by plasma protein concentration. Plasma protein data were not collected in conjunction with blood volume measurements made during the 4th wk of training.

Muscle samples were obtained by biopsy of the vastus lateralis. Samples were divided into several pieces, placed in vials, and stored in liquid nitrogen until they could be freeze-dried and analyzed for citrate synthase activity (26). Citrate synthase values represent the average of measured activity in two pieces. The coefficient of variation between the pieces averaged <2% (of the mean) and in no case was >5%.

**Data analysis.** Multifactor analysis of variance (ANOVA) was used to determine whether there were significant differences between groups and repeated measurements. When the ANOVA indicated that factors (group, training) had significant main or interactive effects, Tukey's critical difference was calculated and used to identify significant differences between means. Simple linear correlation analysis was used to explore possible relationships between selected parameters. A computerized statistical package (CSS:STATISTICA, Statsoft) was used to analyze the data. Data are reported as means  $\pm$  SE. For all statistical procedures, significance was assumed when  $P < 0.05$ .

## RESULTS

**Training program.** Although subjects were encouraged to make up on weekends any training sessions missed during the week, this was not always possible. The actual number of training sessions completed over the 8-wk period ranged from 30 to 39, with no significant difference in the number of sessions completed between the CWT ( $35 \pm 1$ ) and the HWT ( $36 \pm 1$ ). There were no significant changes in body mass or percent fat from before to after training.

Figure 1 shows the  $\dot{V}\text{O}_2$ , final (59th min) rectal temperature, and final heart rate during the exercise training sessions. Throughout the training program, heart rate at the end of exercise averaged 27 beats/min higher ( $P < 0.001$ ) for the HWT than for the CWT. Heart rate at the end of exercise averaged  $\sim 8$  beats/min higher during the 3rd wk compared with the 1st wk of training; final heart

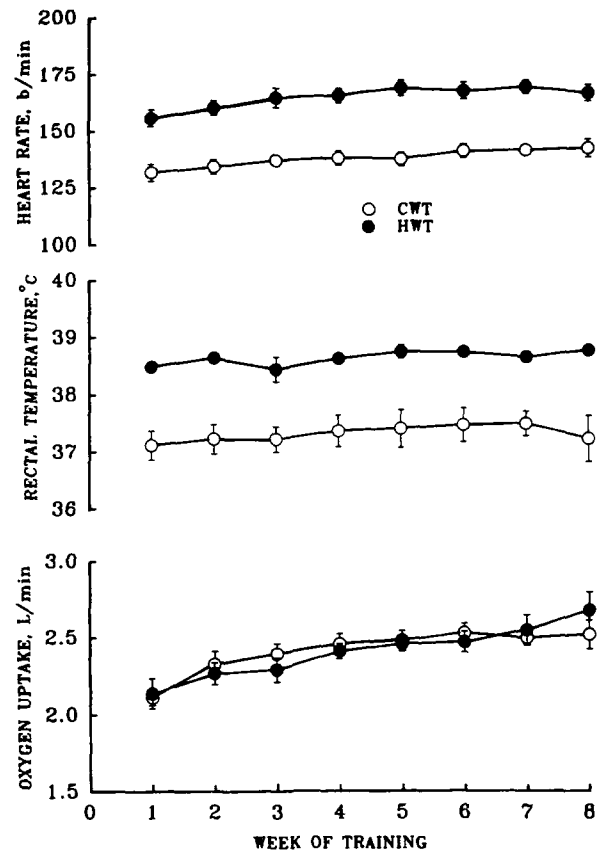


FIG. 1. Heart rate (top), rectal temperature (middle), and oxygen uptake (bottom) of cold (CWT) and hot (HWT) water training groups during exercise each training week. b/min, beats/min.

rates were not significantly changed from the 4th wk to the end of training. Preexercise  $T_{re}$  (not shown) did not differ between the CWT and HWT. Throughout the training program,  $T_{re}$  of the CWT remained unchanged during exercise while the HWT experienced an increase ( $P < 0.01$ ). The final exercise  $T_{re}$  of the HWT was  $\sim 1.5^\circ\text{C}$  higher than that of the CWT. Final exercise  $T_{re}$  during the 5th training wk and thereafter was slightly ( $\sim 0.3^\circ\text{C}$ ,  $P < 0.001$ ) higher than during the 1st training wk. The  $\dot{V}\text{O}_2$  during exercise training increased by  $\sim 400$  ml/min ( $P < 0.0001$ ) progressively over the 8-wk program, but there were no differences between the CWT and HWT in  $\dot{V}\text{O}_2$  during exercise training. Relative exercise intensity ( $\dot{V}\text{O}_2$  expressed as percent  $\dot{V}\text{O}_{2\text{max}}$ ) was the same for the CWT and HWT throughout the training program. Relative exercise intensity averaged  $61 \pm 1\%$  during the 1st training wk, increased slightly ( $P < 0.01$ ) to  $65 \pm 1\%$  during the 4th wk, and remained unchanged for the remainder of the training program.

**Maximal exercise.** Figure 2 shows the effects of endurance training in cold and hot water on  $\dot{V}\text{O}_{2\text{max}}$  of the subjects. Compared with levels measured before training,  $\dot{V}\text{O}_{2\text{max}}$  increased ( $P < 0.01$ ) after 4 wk of training; a further increase ( $P < 0.01$ ) in  $\dot{V}\text{O}_{2\text{max}}$  was experienced between the 4th and 8th wk. Overall, there was a 13% increase in  $\dot{V}\text{O}_{2\text{max}}$  with training, with no significant difference between the CWT and HWT in the magnitude of this effect. There was no significant relationship ( $r =$

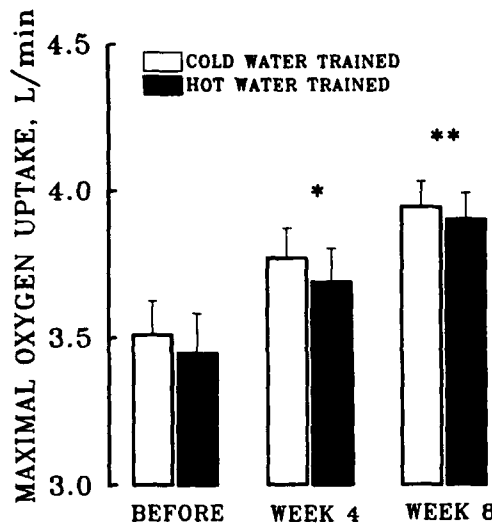


FIG. 2. Maximal oxygen uptake before and after training in cold or hot water. Values are means  $\pm$  SE. \*Significantly different from before training; \*\* significantly different from before training and week 4.

0.17,  $P > 0.5$ ) between initial level of aerobic fitness (as indicated by the  $\dot{V}O_{2\max}$  normalized to body mass) and the training-induced increase in  $\dot{V}O_{2\max}$ .

The maximal heart rates before and after 4 and 8 wk of training are shown in Figure 3. Maximal heart rate did not differ significantly between groups, either before or after training. There was a tendency for maximal heart rate to decline with training, particularly in the HWT, but this was not statistically significant ( $P = 0.07$ ).

**Muscle oxidative capacity.** There were no significant differences in citrate synthase activity of the CWT ( $87 \pm 4 \mu\text{mol} \cdot \text{min}^{-1} \cdot \text{g dry tissue}^{-1}$ ) and HWT ( $88 \pm 4 \mu\text{mol} \cdot \text{min}^{-1} \cdot \text{g}^{-1}$ ) before training. Training resulted in a 38% increase ( $P < 0.001$ ) in citrate synthase activity, with no significant difference between the CWT ( $124 \pm 10 \mu\text{mol} \cdot \text{min}^{-1} \cdot \text{g}^{-1}$ ) and HWT ( $119 \pm 6 \mu\text{mol} \cdot \text{min}^{-1} \cdot \text{g}^{-1}$ ) in the magnitude of the training effect. The increase in citrate synthase activity was correlated ( $r = 0.49$ ,  $P < 0.04$ ) with the increase in aerobic fitness, regardless of training group, as reflected by the increase in  $\dot{V}O_{2\max}$  normalized to body mass.

**Blood volume.** There were no significant differences between the CWT and HWT in erythrocyte, plasma, blood volume, or total circulating protein either before beginning or after completing training. Total circulating protein was unchanged by training, averaging  $230 \pm 6$  g before and  $239 \pm 7$  g after 8 wk of training. The effect of training on vascular volumes is shown in Table 2. Plasma volume was unchanged by training. There were no changes in erythrocyte volume apparent between the 1st and 4th wk of training, but after 8 wk of training, both the CWT and HWT exhibited a small (average for all 18 subjects was  $80 \pm 20$  ml or  $4.9 \pm 1.3\%$  relative to pretraining) but statistically significant ( $P < 0.01$ ) increase in erythrocyte volume. There was no relationship ( $P = 0.378$ ,  $P = 0.12$ ) between the erythrocyte response to training and the pretraining aerobic fitness as reflected by the  $\dot{V}O_{2\max}$  normalized to body mass. Despite the increase in erythrocyte volume, blood volume was not significantly changed.

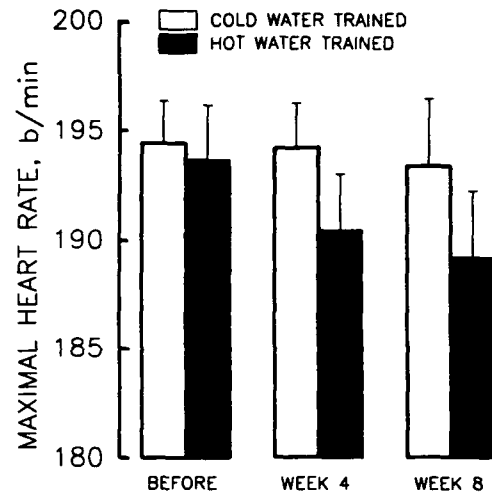


FIG. 3. Maximal heart rate before and after training in hot or cold water. Values are means  $\pm$  SE.

## DISCUSSION

This study is the first to examine the effects of manipulation of body temperature during exercise on adaptations in the physiological determinants of aerobic capacity resulting from endurance training. Avellini et al. (4) reported the effects of manipulating body temperature during exercise training on responses to heat stress. They observed that thermoregulatory adaptations to endurance training differed in subjects who trained in hot water and experienced an increase in body temperature compared with those who trained in cold water and whose body temperature remained unchanged during exercise. Unpublished observations from that study (K. B. Pandolf, personal communication) suggested that improvements in work capacity occurred earlier in subjects exercising in cold than those training in hot water. Combined with the fact that cardiovascular (23) and metabolic (27) responses to exercise in hot environments differ from responses seen in cool environments, these observations suggested the possibility that nonthermoregulatory adaptations to training might also differ when exercise was performed in hot vs. cold water. In addition, others (14) have speculated that the addition of thermal stress during exercise might alter the stimulus for training adaptations.

However, the results of this investigation indicate that thermal factors are not important for nonthermoregulatory adaptations to aerobic training. After 8 wk of training,  $\dot{V}O_{2\max}$ , muscle citrate synthase activity, and erythrocyte volume were all significantly increased, but the magnitude of the training effects was the same for the HWT and CWT. Before training began, the two groups were matched closely for age, body composition, and initial level of aerobic fitness. Both groups complied well with the prescribed exercise regimen and, except for the very different body temperatures achieved during exercise, they trained identically, as indicated by the similar  $\dot{V}O_2$  levels during training. Although the data suggest the possibility that training in hot water might cause maximal heart rate to fall, overall, it appears that the rise in body temperature during exercise does not contribute

TABLE 2. *Effect of training in cold or hot water on vascular volumes*

|        | Erythrocyte Volume |          |           | Plasma Volume |           |           | Blood Volume |           |           |
|--------|--------------------|----------|-----------|---------------|-----------|-----------|--------------|-----------|-----------|
|        | Pre                | Mid      | Post      | Pre           | Mid       | Post      | Pre          | Mid       | Post      |
| CWT    | 1,878±101          | 1,885±77 | 1,936±72* | 3,275±126     | 3,244±165 | 3,307±109 | 5,153±220    | 5,240±158 | 5,240±149 |
| HWT    | 1,849±63           | 1,897±60 | 1,955±52* | 3,162±111     | 3,222±139 | 3,222±169 | 5,011±161    | 5,117±193 | 5,178±169 |
| Pooled | 1,864±58           | 1,891±47 | 1,945±43* | 3,219±82      | 3,233±106 | 3,264±84  | 5,082±133    | 5,178±122 | 5,210±110 |

Values are means  $\pm$  SE for 9 men in both CWT and HWT. Measurements were made before (Pre) and during 4th (Mid) and 8th (Post) wk of endurance training in cold (CWT) or hot (HWT) water. \* Significantly different from pretraining.

importantly to the stimulus for aerobic adaptations during endurance training.

This longitudinal investigation demonstrates that blood volume expansion is not a prerequisite for an increase in aerobic fitness resulting from endurance training. Despite the substantial increase ( $\sim 13\%$ ) in  $\dot{V}O_{2\max}$ , blood volume remained unchanged with 8 wk of training in either hot or cold water. Although this observation agrees with findings of a recently published cross-sectional study (24) in which no relationship between aerobic fitness and blood volume was apparent, it conflicts with the view expressed by Convertino (7) that hypervolemia is an important consequence of endurance training. In support of that view, Convertino presented a regression analysis of cross-sectional data, which demonstrated a correlation between blood volume and aerobic fitness. The regression equation Convertino derived predicts that people having a  $\dot{V}O_{2\max}$  in the range encompassed by the subjects of the present investigation should have had a blood volume of  $75\text{--}83\text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , which is considerably larger than that actually measured.

Blood volume of the subjects in this study averaged  $\sim 69\text{ ml/kg}$  body wt, and  $\dot{V}O_{2\max}$  averaged  $47\text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  (pretraining) and  $52\text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  (posttraining). These values are very similar to those reported by other investigators using radioactively labeled red blood cells (22, 24). Convertino (7) did not specify the methodology used to obtain blood volume values for the regression analysis. However, the most widely used methodologies of determining blood volume, estimating erythrocyte volume from plasma volume measured by Evans blue and measuring erythrocyte volume using the carbon monoxide technique, both result in overestimates because the volume of distribution for both of those tracers is not strictly limited to the vascular space (5, 20).

An increase in plasma volume has been observed in many endurance-training studies (7, 10, 11, 20). In studies employing training programs lasting  $<10$  days and demonstrating an increase in blood volume, the change in blood volume can be completely accounted for by an increase in plasma volume (7). Furthermore, an increase in plasma volume has been demonstrated after as little as one training session, when the training intensity was very severe (11). However, in the present investigation, plasma volume was not significantly changed by 8 wk of endurance training. Perhaps the training intensity used in this investigation was not sufficiently high to stimulate an increase in plasma volume, or possibly an increase in plasma volume is an early and transient response to training, in which case the plasma volume measurements made during this study were not at the correct

time to demonstrate an increase. On the other hand, increases in plasma volume have been observed in studies (10, 20) employing longer training periods and/or exercise training intensities comparable to those used in this investigation.

One further possibility is that the stimulus for an increase in plasma volume and hypervolemia with training was not present because the exercise was performed in water. Perrault et al. (21) reported that release of arginine vasopressin, renin, and aldosterone is suppressed during supine compared with upright exercise, whereas release of atrial natriuretic peptide is enhanced. Presumably, these effects are mediated by facilitated venous return during supine compared with upright exercise, which in turn would result in atrial distension and release of natriuretic peptide and cardiopulmonary baroreceptor-stimulated suppression of the renin-aldosterone axis. These posture-related differences in fluid-regulatory hormone responses to exercise would be expected to contribute to differences in plasma volume responses to training by upright vs. supine exercise, and, indeed, Ray et al. (22) observed that plasma volume increased with training by upright but not supine exercise. Upright immersion results in hemodynamic and fluid-regulatory hormonal changes similar to those moving from upright to supine posture on land as a result of the hydrostatic pressure gradient that develops between the lower limbs and the central thorax (9). Therefore, responses to upright exercise in water appear to be similar to those during supine exercise on land.

After 8 wk of training, a 4% increase in erythrocyte volume was apparent compared with before training. This finding agrees with those of others who have observed modest increases in erythrocyte volume associated with endurance training and improved aerobic fitness (22). Although changes in erythrocyte volume were not statistically significant after 4 wk of training, inspection of Table 2 does suggest that this response to training is progressive over time. Perhaps more significant enhancement of erythrocyte volume could be demonstrated in longitudinal studies employing training periods longer than 8 wk, in which case improved aerobic capacity may indeed be shown to be related to increased erythrocyte volume.

The results of this study indicated that sustaining different heart rates during training had no effect on aerobic training adaptations. The two groups achieved equivalent increases in  $\dot{V}O_{2\max}$ , muscle citrate synthase, and erythrocyte volume despite the fact that the heart rate during exercise training was  $>25$  beats/min lower for the CWT than HWT. Although interesting, this observation

is readily explainable. It is well known that when body temperatures are elevated during exercise, higher heart rates will be achieved than when body temperatures are lower (23). However, it is the exercise intensity that determines the magnitude of overload and associated training stimulus. In this study, the training intensity was the same for both groups, as demonstrated by the comparable levels of  $\dot{V}O_2$ , percent  $\dot{V}O_{2\max}$ , and, presumably, cardiac output (17) during exercise. This observation may have implications for fitness programs in which a target heart rate is prescribed for individuals to achieve during training, particularly programs employing aquatic exercise.

In summary, this study demonstrated that exercise-induced increases in body temperature are not an important stimulus for aerobic capacity adaptations to endurance training. Additionally, hypervolemia is not a prerequisite for improved aerobic capacity associated with endurance training, and endurance training in water does not produce the expansion of plasma volume and hypervolemia often observed to occur during endurance training on land.

We gratefully acknowledge the assistance of James Bogart, Penny Burgoon, Dana Cohen, William Latzka, Janet Laird, Leslie Levine, Stephen Muza, Linda Pivacek, and Dan Schlatterer in the collection and analyses of the data contained in this report. We also thank the soldiers who volunteered to participate as subjects in this investigation, because without their efforts the study would not have been accomplished.

The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as an official Department of the Army or Navy position, policy, or decisions, unless so designated by other official documentation. Approved for public release; distribution is unlimited.

Address for reprint requests: A. J. Young, Thermal Physiology and Medicine Division, US Army Research Institute of Environmental Medicine, Natick, MA 01760-5007.

Received 10 September 1992; accepted in final form 10 February 1993.

## REFERENCES

- ADAMS, T., AND E. J. HEBERLING. Human physiological responses to a standardized cold stress as modified by physical fitness. *J. Appl. Physiol.* 13: 226-230, 1958.
- ANDERSEN, K. L. Metabolic and circulatory aspects of tolerance to cold as affected by physical training. *Federation Proc.* 25: 1351-1356, 1966.
- ARMSTRONG, L. E., AND K. B. PANDOLF. Physical training, cardiorespiratory physical fitness and exercise-heat tolerance. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, edited by K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis, IN: Benchmark, 1988, p. 199-226.
- AVELLINI, B. A., Y. SHAPIRO, S. M. FORTNEY, C. B. WENGER, AND K. B. PANDOLF. Effects on heat tolerance of physical training in water and on land. *J. Appl. Physiol.* 53: 1291-1298, 1982.
- BASS, D. E., E. R. BUSKIRK, P. F. IAMPINETRO, AND M. MAGER. Comparison of blood volume during physical conditioning, heat acclimatization and sedentary living. *J. Appl. Physiol.* 12: 186-188, 1958.
- BITTEL, J. H. M., C. NONOTT-VARLY, G. H. LIVECCHI-GONNOT, G. L. M. J. SAVOUREY, AND A. M. HANNIQUET. Physical fitness and thermoregulatory reactions in a cold environment in men. *J. Appl. Physiol.* 65: 1984-1989, 1988.
- CONVERTINO, V. A. Blood volume: its adaptation to endurance training. *Med. Sci. Sports Exercise* 23: 1338-1348, 1992.
- CONVERTINO, V. A., J. E. GREENLEAF, AND E. M. BERNAUER. Role of thermal and exercise factors in the mechanism of hypervolemia. *J. Appl. Physiol.* 48: 657-664, 1980.
- EPSTEIN, M. Cardiovascular and renal effects of head-out water immersion in man: application of the model in the assessment of volume homeostasis. *Circ. Res.* 39: 619-628, 1976.
- FORTNEY, S. M., AND L. C. SENAY. Effect of training and heat acclimation on exercise responses of sedentary females. *J. Appl. Physiol.* 47: 978-984, 1979.
- GREEN, H. J., J. A. THOMSON, M. E. BALL, R. L. HUGHSON, M. E. HOUSTON, AND M. T. SHARRATT. Alterations in blood volume following short-term supramaximal exercise. *J. Appl. Physiol.* 56: 145-149, 1984.
- HOLLOSZY, J. O., AND E. F. COYLE. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J. Appl. Physiol.* 56: 831-838, 1984.
- HOLMER, I., AND U. BERGH. Metabolic and thermal response to swimming in water at varying temperatures. *J. Appl. Physiol.* 37: 702-705, 1974.
- HOUSTON, M. E., N. J. CHRISTENSEN, H. GALBO, J. J. HOLST, B. NIELSEN, E. NYGAARD, AND B. SALTIN. Metabolic responses to swimming at three different water temperatures. In: *Swimming Medicine IV*, edited by B. Eriksson, B. Furberg, R. C. Nelson, and C. A. Moorehouse. Baltimore, MD: University Park Press, 1978, p. 327-333.
- KAMON, E., AND K. B. PANDOLF. Maximal aerobic power during laddermill climbing, uphill running, and cycling. *J. Appl. Physiol.* 32: 467-473, 1972.
- KOLLIAS, R. B., AND E. R. BUSKIRK. Effects of physical condition in man on thermal responses to cold air. *Int. J. Biometeorol.* 16: 389-402, 1972.
- MCARDLE, W. D., J. R. MAGEL, G. R. LESMES, AND G. S. PECHAR. Metabolic and cardiovascular adjustment to work in air and water at 18, 25, and 33°C. *J. Appl. Physiol.* 40: 85-90, 1976.
- MOORE, L. G., A. CYMERMAN, H. SHAO-YUNG, R. E. MCCULLOUGH, R. G. MCCULLOUGH, P. B. ROCK, A. YOUNG, P. M. YOUNG, D. BLOEDOW, J. V. WEIL, AND J. T. REEVES. Propranolol does not impair exercise oxygen uptake in normal men at high altitude. *J. Appl. Physiol.* 61: 1935-1941, 1986.
- NADEL, E. R., I. HOLMER, U. BERGH, P. O. ASTRAND, AND J. A. J. STOLWIJK. Energy exchanges of swimming man. *J. Appl. Physiol.* 36: 465-471, 1974.
- OSCAI, L. B., B. T. WILLIAMS, AND B. A. HERTIG. Effect of exercise on blood volume. *J. Appl. Physiol.* 24: 622-624, 1968.
- PERRAULT, H., M. CANTIN, G. THIRIAULT, G. R. BRISSON, AND M. BELAND. Plasma atrial natriuretic peptide during brief upright and supine exercise in humans. *J. Appl. Physiol.* 66: 2159-2167, 1989.
- RAY, C. A., K. J. CURETON, AND H. G. OUZTS. Postural specificity of cardiovascular adaptations to exercise training. *J. Appl. Physiol.* 69: 2204-2208, 1990.
- SAWKA, M. N., AND C. B. WENGER. Physiological responses to acute exercise-heat stress. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, edited by K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis, IN: Benchmark, 1988, p. 97-151.
- SAWKA, M. N., A. J. YOUNG, K. B. PANDOLF, R. C. DENNIS, AND C. R. VALERI. Erythrocyte, plasma, and blood volume of healthy young men. *Med. Sci. Sports Exercise* 24: 447-453, 1992.
- SHAPIRO, Y., B. A. AVELLINI, M. M. TONER, AND K. B. PANDOLF. Modification of the Monark bicycle ergometer for underwater exercise. *J. Appl. Physiol.* 50: 679-683, 1981.
- SRERE, P. A. Citrate synthase. *Methods Enzymol.* 13: 3-11, 1969.
- YOUNG, A. J. Energy substrate utilization during exercise in extreme environments. *Exercise Sport Sci. Rev.* 18: 65-117, 1990.

DTIC QUALITY INSPECTED 3

| Availability Codes |                      |
|--------------------|----------------------|
| Dist               | Avail and/or Special |
| A-1                | 20                   |